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## Treatment and Management of Common Endocrine Disorders

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### Review Article

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#### ABSTRACT

Endocrine glands are the major glands which have multiple functions. Increased or decrease in the secretion of endocrine glands leads to disorders in the endocrine system. Major Glands such as Pituitary, Thyroid, Pancreas and Gonads were studied. Deficiency or Hypersecretion of these glands results in the disorder of the endocrine glands. Treatment for the endocrine disorders can be done by Molecular diagnosis and Imaging. The Complications in the system can be avoided by preventing the risk factors causing the abnormality. Present review discusses on the Study of endocrine system, Disorders in the system, Abnormalities, Complications, Treatment and Management of endocrine disorders.

#### INTRODUCTION

Endocrine glands are the glands of endocrine system which normally secrete special products called Hormones directly into the blood [1-4]. They do not have ducts and hence they are called as ductless glands. Endocrine glands are subdivided into three groups namely Endocrine gland hypo secretion (less secretion of hormones), Endocrine gland hypersecretion (More secretion of hormones) and Tumours of endocrine glands [5,7,9]. Hormonal Disorders are difficult to diagnose as they cannot be assayed directly from the blood, however indirect methods are helpful [8,10].

#### UNDERSTANDING THE FUNCTIONING OF ENDOCRINE GLANDS

##### Hyperfunction

Hyperfunction of endocrine glands may result from overstimulation by the pituitary but is most commonly due to hyperplasia or neoplasia of the gland itself [11-14]. In some cases, cancers from other tissues can produce hormones (ectopic hormone production) [15]. Hormone excess also can result from exogenous hormone administration. In some cases, patients take hormones without telling the physician (factitious disease). Tissue hypersensitivity to hormones can occur [16,17]. Antibodies can stimulate peripheral endocrine glands, as occurs in hyperthyroidism of Graves' disease. Destruction of a peripheral endocrine gland can rapidly release stored hormone (eg, thyroid hormones in thyroiditis). Enzyme defects in the synthesis of a peripheral endocrine hormone can result in overproduction of hormones proximal to the block [18-20]. Finally, overproduction of a hormone can occur as an appropriate response to a disease state [21,22].

##### Hypofunction

Hypofunction of an endocrine gland can result from understimulation by the pituitary. Hypofunction originating within the peripheral gland itself can result from congenital or acquired disorders (including

autoimmune disorders, tumors, infections, vascular disorders, and toxins). Genetic disorders causing hypofunction can result from deletion of a gene or by production of an abnormal hormone [23-25]. A decrease in hormone production by the peripheral endocrine gland with a resulting increase in production of pituitary regulating hormone can lead to peripheral endocrine gland hyperplasia [26,27]. For example, if synthesis of thyroid hormone is defective, thyroid-stimulating hormone (TSH) is produced in excessive amounts, causing goiter[28-32].

Several hormones require conversion to an active form after secretion from the peripheral endocrine gland. Certain disorders can block this step (eg, renal disease can inhibit production of the active form of vitamin D). Antibodies to the circulating hormone or its receptor can block the ability of the hormone to bind to its receptor[33-35]. Disease or drugs can cause increased rate of clearance of hormones. Circulating substances may also block the function of hormones[36,37]. Abnormalities of the receptor or elsewhere in the peripheral endocrine tissue can also cause hypofunction [38-40].

## ENDOCRINE GLANDS

Some of the endocrine glands include Pituitary gland, Thyroid Gland, Adrenal Gland, Pancreas and Gonads.

### Pituitary Gland

It is the Master gland of the body. It mainly regulates the neurological system and hence it is a neuroendocrine gland. They are responsible for proper maintenance and functioning of the body [39]. Common Disorders of Pituitary gland are Acromegaly, Diabetes Insipidus and Pituitary hormone.

### Acromegaly:

Caused by the excessive secretion of growth hormones. The disease is characterised by Swollen hands, legs and tissues in the body. Symptoms include Swelling of hands and legs, Enlargement of jaw, Degenerative arthritis and enlargement of bones in the body [41-44].

### Diabetes Insipidus:

The disease is characterized by excessive thirst and extreme amount of urine formation. The disease is caused by insufficient production of antidiuretic hormone[ 45,46].

### Hypopituitarism:

It leads to impaired attention and memory, decreased muscle mass and retardation in growth.

### Pituitary Tumours:

The most common pituitary tumours are Adenoma and Prolactinoma

**Adenoma:** They occur commonly in Pituitary gland.

**Prolactinoma:** They secrete prolactin and they are responsible for galactorrhoea, infertility and impotence [47-50].

## THYROID GLAND

The largest of all the endocrine glands is Thyroid Gland. It secretes hormones namely Thyroid helpful for physical and mental development [51,52].

The major disorders of thyroid gland include Hyperthyroidism, Hypothyroidism, Goitre and Thyroid cancer.

**Cretinism:** The condition of severe stunted physical and mental growth is called cretinism [53]. Retarded Skeletal growth, Reduced BMR are the common symptoms of the disease.

**Myxedema:** It is caused by reduced production of thyroxin. Rate of metabolism is reduced to half.

**Graves disease:** It is an auto immune disease where the amount of thyroid is more and overactive.

**Goitre:** The swelling in the thyroid gland results in goitre. The most common cause is iodine deficiency.

**Adrenal Gland:** It secretes hormones such as Aldosterone, Corticosterone and Androgens.

**Pancreas:** It is the second largest gland. Hormones secreted are Insulin, Glucagon, Somatostatin etc.

The major disorders are diabetes mellitus, Hypoglycemia and Pancreatitis.

**Type I Diabetes:**

Type I Diabetes results from body failure to produce insulin and hence the patient is allowed to produce or inject insulin<sup>[54,55]</sup>.

Type II Diabetes results from insulin resistance sometimes with an absolute insulin deficiency.

Gestational diabetes is the type of diabetes that occurs in women during pregnancy.

## GONADS

Gonads include testis and ovaries.

**Ovary:** Ovary is the female reproductive organ that produces ovum. Ovaries secrete both estrogen and progesterone.

Abnormality in the function of ovaries results in the following deficiencies namely

**Ammenorrhoea:** Absence of menstrual blood in reproductive age. It is of two types namely:

**Primary Ammenorrhoea:** It is caused usually due to the congenital absence of uterus or failure of ovary to receive eggs.

**Secondary Ammenorrhoea:** It is caused due to the hormonal disturbances from pituitary and hypothalamus <sup>[56]</sup>.

**Hypogonadism:** This is caused by low levels of estrogen.

**Polycystic Ovarian Syndrome:** It is caused to approximately 5-10% of the women with reproductive age. Obesity, diabetes and insulin resistance are strongly related with PCOS. The ovaries make follicles but the eggs do not mature and leave the ovaries. The immature follicles can turn into fluid filled sacs called cysts. Most women with PCOS have cysts but women with cysts do not have PCOS.

**Abnormalities in Testis functioning :**

The abnormalities in testis include Hypogonadism, Testicular Cancer

**Hypogonadism:** Hypogonadism is a disorder that occurs in men with insufficient production of testosterone. Primary hypogonadism occurs when there is a problem with the pituitary gland which sends chemical messages to the testicles to produce testosterone<sup>[57,58]</sup>.

**Testicular Cancer:** It occurs when abnormal cells in the testicles divide and grow uncontrolled. It can develop in the testicles of younger boys. A lump or irregularity in the testis is the major symptom of testicular cancer<sup>[57-59]</sup>.

## TREATMENT OF ENDOCRINE DISORDERS

In many cases, endocrine disorders may be symptomless or mild enough to not require treatment. Symptoms can arise from excess hormone production or a hormone deficiency. When symptoms of endocrine disorders are bothersome, they can generally be treated by correcting the hormone imbalance. This is often done by means of synthetic hormone administration. In cases such as prolactinoma, where a noncancerous tumor is responsible for symptoms, surgery or radiation therapy may be used. Often, diagnosis and treatment of the underlying cause of the endocrine disorder will resolve the symptoms.

**Complications of Endocrine System:**

While most endocrine disorders are mild and slow to progress, certain endocrine disorders can lead to complications over time as unbalanced hormonal signaling affects normal body processes. In cases of Addison's disease and hypothyroidism in particular, acute attacks or crises can have serious complications <sup>[60]</sup>. Diabetes can also have potentially life-threatening complications. Complications of untreated or poorly controlled endocrine disorders can be serious, even life threatening in some cases.

**Risk factors of Endocrine Disorders:**

A number of factors increase the risk of developing endocrine disorders. Not all people with risk factors will develop endocrine disorders. Risk factors for endocrine disorders include: Elevated cholesterol levels, Family history of endocrine disorder, Inactivity, Personal history of autoimmune disorders, such as diabetes, Poor diet, Pregnancy (in cases such as hyperthyroidism), Recent surgery, trauma, infection, or serious injury<sup>[61]</sup>.

#### **Advances in treatment of Endocrine Disorders:**

Although the investigations relate to rare single disorders, they have resulted in major breakthroughs in many fields of endocrinology contributing to the understanding of cellular mechanism of hormone action.

#### **Molecular Investigation and Diagnosis:**

The tools used to treat the disease is mainly by molecular diagnostic techniques. The diagnosis of insulin hormone levels and G Protein Couple Receptors can be done by molecular diagnosis<sup>[62]</sup>.

### **CONCLUSION**

Obesity is an important contributor, not only to diabetes risk in minority populations, but also to sex disparities in thyroid cancer, which is more common in women. This suggests that population interventions targeting weight loss may have a favorable impact, even on non-diabetes-related endocrine disorders. Our statement also highlights important implications regarding the definition of obesity in different race/ethnic groups <sup>[59-61]</sup>. Fat distribution is an important determinant of risk of metabolic disorders such as type 2 diabetes and CVD, and there is evidence that current cut-points for WC may underestimate disease risk in Asian-Americans and over estimate disease risk on NHB women<sup>[63]</sup>. Specific treatment still doesn't exist for the treatment of hormonal disorders. Avoiding risk factors is the major important tool for the treatment of the disease <sup>[63,64]</sup>.

### **REFERENCES**

1. Alvina R Kansra Infantile Hypophosphatasia: An unusual Presentation and Novel Gene Mutation. *Adv Genet Eng* 2015; 3:102.
2. Strifert K The Case for Epidemiological Investigation of the Possible Link between Combined Oral Contraceptives and Autism Spectrum Disorder. *Endocrinol Metab Synd* 2015; 4:165.
3. Speiser PW Adrenal Dysfunction. *Endocrinol Metab Synd* 2015; 4:164.
4. Vélayoudom-Céphise F, Foucan L, Larifla L, Chingan V, Bangou J, et al. Relationship between Testosterone and Sex Hormone Binding Globulin Concentrations with Cardiometabolic Parameters and Macrovascular Disease in Afro-Caribbean Men with Type 2 Diabetes. *Endocrinol Metab Synd* 2015; 4:162.
5. Sechi LA, Colussi GL, Catena C Role of Aldosterone in Insulin Resistance: Fact of Fantasy. *Endocrinol Metab Synd* 2015;4:161.
6. Chakraborty PP, Chowdhury S A Look Inside the Pancreas: The "Endocrine-Exocrine Cross-talk". *Endocrinol Metab Synd* 2015; 4:160.
7. Shalaby A, Eliwa KAA, Hassan AM, El-Fiky M Sex Differences in Some Physiological Effects of Cold Season or Short-Term Cold Exposure in Adult Albino Rat. *Endocrinol Metab Synd* 2015; 4:159.
8. Hackeny AC, Lane AR Exercise Endocrinology: Guidance for Future Research Direction and Focus. *J Steroids Hormon Sci* 2015; 6:e114.
9. Vicennati V, Pinna AD, Morelli MC, Pagotto U, Pasquali R Prevalence of Metabolic Syndrome in Organ Transplantation: A Review of the Literature. *Endocrinol Metab Synd* 2015; 4:157.

10. Valeska AC, Carina AV, Paula P, Ana MR, Miguel NL Stress during Lactation Affects Fatty Acid Amide Hydrolase Protein Expression in Adipose Tissue and Liver of Adult Mice. *Endocrinol Metab Synd* 2015; 4:158.
11. Bohra A, Bhateja S Carcinogenesis and Sex Hormones: A Review. *Endocrinol Metab Synd* 2015; 4:156.
12. Huang W Influences of Gut Hormones on Hepatocellular Carcinoma. *Endocrinol Metab Synd* 2015; 4:155.
13. Pirasath S Glycemic Index of Traditional Foods in Northern Sri Lanka. *Endocrinol Metab Synd* 2015; 4:154.
14. Nikkhah A Intake Circadian Physiology: An Overlooked Public Health Concern. *Endocrinol Metab Synd* 2014; 4:153.
15. Stoll H, Hamel FG, Lee JS, Ha L, Lim JY (Mechanical Control of Mesenchymal Stem Cell Adipogenesis. *Endocrinol Metab Synd* 2015;4:152.
16. Mohammed Differentiation between the Anterior Pituitary Cells of the Egyptian Insectivorous Bats *Rhinopoma hardwickei* using Transmission Electron Microscope. *Endocrinol Metab Synd* 2015; 4:151.
17. Carrillo LFM Negative Urinary Fractionated Metanephrines and Elevated Urinary Vanillylmandelic Acid in a Patient with a Sympathetic Paravesical Paraganglioma. *Endocrinol Metab Synd* 2014; 4: i004.
18. Dodani S Coronary Artery Diseases in South Asian Immigrants: Philosophy behind HDL Function. *Endocrinol Metab Synd* 2015;4:e123.
19. Mpora OB, Oliver E, Barbara A, Francis O, Wilfred W, et al. Glucose Addiction and Glycemic Control in Type 2 Diabetes Mellitus: A Case Report. *Endocrinol Metab Synd* 2014; 3:150.
20. León-Toirac EJ Immune Network, the Dangerous Liaisons in Pain: A Short Review. *J Pain Relief* 2015; 4:170.
21. Shwayhat AF, Hoang TD, Poremba JA, Acosta RD, Shakir MKM The Effect of Exercise on 1 mcg ACTH Stimulation of the Hypothalamic-Pituitary-Adrenal Axis. *Endocrinol Metab Synd* 2014; 3:148.
22. Habtewold TD, Mulugeta S, Gebreegziabhere Y A Cross Sectional Study on Associated Factors of Depression among Type 2 Diabetic Outpatients in Black Lion General Specialized Hospital, Addis Ababa, Ethiopia. *Endocrinol Metab Synd* 2014; 3:147.
23. Kamenova P, Atanasova I, Kirilov G Metformin Reduces Cardiometabolic Risk Factors in People at High Risk for Development of Type 2 Diabetes and Cardiovascular Disease. *J Diabetes Metab* 2014; 5:470.
24. Guénard F, Cormier M, Tchernof A, Deshaies Y, Biron S, et al. Common Sequence Variants in CD163 Gene are Associated with Plasma Triglyceride and Total Cholesterol Levels in Severely Obese Individuals. *Endocrinol Metab Synd* 2014; 3:146.
25. Olsen-Bergem H, Bjørnland T, Reseland JE Temporomandibular Joint Pain is Negatively Correlated to TNF Alpha and Osteoprotegerin Content in Synovial Fluid in Patients with Juvenile Idiopathic Arthritis. *Endocrinol Metab Synd* 2014; 3:145.
26. Woldu MA, Wami CD, Lenjisa JL, Tegegne GT, Tesafye G, et al. Factors Associated with Poor Glycemic Control among Patients with Type 2 Diabetes Mellitus in Ambo Hospital, Ambo; Ethiopia. *Endocrinol Metab Synd* 2014; 3:143.
27. Tan M, Kim SH Does Polycystic Ovarian Syndrome Increase Insulin Resistance Above and Beyond Obesity?. *Endocrinol Metab Synd* 2014; 3:142.
28. Genazzani AD, Despini G, Santagni S, Prati A, Rattighieri E, et al. Effects of a Combination of Alpha Lipoic Acid and Myo-Inositol on Insulin Dynamics in Overweight/Obese Patients with PCOS. *Endocrinol Metab Syndr* 2014; 3:140.

29. KoÅš ZP, Temelli B, KiliÅš L, Simsek FS Transfection with Sodium Iodine Symporter Gene (NIS) and Future Applications with Radioiodine Treatment. *Endocrinol Metab Syndr* 2014; 3:139.
30. Ramin C, Barrett HL, Callaway LK, Nitert MD The Role of Irisin in Gestational Diabetes Mellitus: A Review. *Endocrinol Metab Syndr* 2014; 3:138.
31. Wahl R, Horger M, Haap M Sublingual Thyroid Gland. *Endocrinol Metab Syndr* 2014;3:i001.
32. Ntentie FR, Ngondi JL, Azantsa KBG, Santy EV, Dimodi HT, et al. Urbanization and Metabolic Syndrome in Cameroon: Alertness on Less Urbanised Areas. *Endocrinol Metab Syndr* 2014; 3:137.
33. Unfer V, Proietti S, Gullo G, Porcaro G, Carlomagno G, et al. Polycystic Ovary Syndrome: Features, Diagnostic Criteria and Treatments. *Endocrinol Metab Syndr* 2014; 3:136.
34. de Piano A, Estadella D, Oyama LM, Ribeiro EB, DÃ¢maso AR, et al. Nonalcoholic Fatty Liver Disease (NAFLD), a Manifestation of the Metabolic Syndrome: New Perspectives on the Nutritional Therapy. *Endocrinol Metab Syndr* 2014; 3:135.
35. Comhaire F Nutraceutical Approach to the Metabolic Syndrome. *Endocrinol Metab Syndr* 2014; 3:134.
36. Balkan F, Usluogullari CA, Ucler R, Baser H, Ersoy R, et al. Does Increased Body Mass Index Lead to Elevated Thyroid Cancer Risk?. *Endocrinol Metab Syndr* 2014; 2:132.
37. Xu Q, Guo S, Xiao X Recent Progress in Genetic Polymorphisms and Diabetic Retinopathy (DR) in Type 1 Diabetes Mellitus (T1DM). *Endocrinol Metab Syndr* 2014; 3:131.
38. Oguejiofor O, Odenigbo C, Onwukwe C Diabetes in Nigeria: Impact, Challenges, Future Directions. *Endocrinol Metab Syndr* 2014; 3:130.
39. Abdalla B and Bishara B Nutrition Style and Diet Composition Leading to Obesity and Overweight in a Cross-Sectional Survey Conducted in a Hospitalized Arab Population in Nazareth, Israel. *Endocrinol Metab Syndr* 2014; 3:129.
40. Obembe OO, Olopade JO Raji Y Implication of Hongres1 Protein in Quassin-Induced Male Reproductive Abnormality in Rats. *Endocrinol Metab Syndr* 2014; 3:128.
41. do Valle Couto Reis V, de Oliveira Coelho GM, de Abreu Soares E, Pereira AF Effect of Dietetic Intervention in Brazilian Postmenopausal Women with Metabolic Syndrome. *Endocrinol Metab Syndr* 2014; 3:127.
42. Sagsak E, Onder A, Ocal FD, Tasci Y, Agladioglu SY, et al. Primary Amenorrhea Secondary to Mullerian Anomaly. *J Clin Case Rep* 2014; S1:007.
43. Biesenbach G, Pieringer H Onset of End-Stage Renal Disease and Prevalence of Vascular Diseases at the Start of Dialysis in Type 2 Diabetic Patients with Diabetic and Vascular Nephropathy. *Endocrinol Metab Syndr* 2014; 3:124.
44. Deghima S, Yahiaoui S, Boudiba A Position of Incretin Agents in the Traitement of Type 2 Diabetes Mellitus: Literature Review. *Endocrinol Metab Syndr* 2014; 3: 123.
45. Botushanov N, Yaneva M, Orbetzova M, Botushanova A Bone Mineral Density in Bulgarian Patients with Type 1 Diabetes Mellitus. *J Osteopor Phys Act* 2014; 2: 113.
46. Mohamed WS, Hassanien MA, Sayed Abokhosheim KEL Role of Ghrelin, Leptin and Insulin Resistance in Development of Metabolic Syndrome in Obese Patients. *Endocrinol Metab Syndr* 2014; 3: 122.
47. Rocha VN, Ferreira RN, Mandarim-de-Lacerda CA, de Carvalho JJ Beneficial Effects of Rosuvastatin in Heart of C57Bl/6 Mice with Diet-Induced Metabolic Syndrome - A Preliminary Study. *Endocrinol Metab Syndr* 2014; 3: 121.
48. Rezq AM (2014) Curcumin Derivatives in Experimental Diabetes. *Endocrinol Metab Syndr* 3:120.
49. Kohno M, Tajima O, Uezono K, Tabata S, Abe H, et al. Cytochrome P450 1A2 Polymorphisms, Coffee Consumption and Impaired Glucose Metabolism in Japanese Men. *Endocrinol Metab Syndr* 2013; 2: 119.

50. Laganà AS, Pizzo A Know your Enemy: The Rationale of Using Inositol in the Treatment of Polycystic Ovary Syndrome. *Endocrinol Metab Synd* 2013; 2: e121.
51. Hurwitz BE Multifaceted Lifestyle Interventions and Cardiometabolic Outcomes in Type 2 Diabetes Mellitus. *Endocrinol Metab Synd* 2013; 2: e120.
52. Zaman F, Faisal Ahmed S, Ward LM Glucocorticoids Action in Bone and Cartilage: A Report from 9th Joint Meeting of Pediatric Endocrinology. *J Steroids Horm Sci* 2014; 5: 120.
53. Peppas M, Koliaki C, Dimitriadis G Body Composition as an Important Determinant of Metabolic Syndrome in Postmenopausal Women. *Endocrinol Metabol Syndrome* 2012; S1: 009.
54. Silveira LS, Buonani C, Monteiro PA, Mello Antunes BM, Freitas JÃºnior IF Metabolic Syndrome: Criteria for Diagnosing in Children and Adolescents. *Endocrinol Metab Synd* 2013; 2:118.
55. Haidara M Stem Cell as a Novel Therapy for Diabetic Cardiomyopathy. *Endocrinol Metab Synd* 2013; 2: e119.
56. Bomfim GF, Szasz T, Carvalho MHC, Webb RC The Toll way to hypertension: role of the innate immune response. *Endocrinol Metab Synd* 2011; 2: 117.
57. Buechler C Chemerin in Non-Alcoholic Fatty Liver Disease â€œ Up or Down? *Endocrinol Metab Synd* 2013; 2: e117.
58. Shpakov AO GPCR-Peptides: Prospective Use in Biology and Medicine. *Endocrinol Metab Synd* 2013; 2: e116.
59. Bandaru P, Rajkumar H, Nappanveetil G The Impact of Obesity on Immune Response to Infection and Vaccine: An Insight into Plausible Mechanisms. *Endocrinol Metab Synd* 2013; 2: 113.
60. Granados H, Phulwani P Absent Visualization of a Hypoplastic Uterus in a 16 Year Old with Complete 46 XY Gonadal Dysgenesis (Swyer Syndrome). *Endocrinol Metab Synd* 2013; 2: 114.
61. Dušková M, Pospíšilová H, Hill M, Stárka L Obesity, Circulating Androgens and their Precursors. *J Steroids Horm Sci* 2013; 4: 119.
62. SatuÅ© K, GardÃ³n JC A Review of the Estrous Cycle and the Neuroendocrine Mechanisms in the Mare. *J Steroids Horm Sci* 2013; 4: 115.
63. Govindan J, Singh I, Kamath C, Gleeson A, Adlan MA, et al. Severe Refractory Hypoglycaemia in an Acutely Ill Elderly Man with Type 2 Diabetes Mellitus. *J Diabetes Metab* 2013; 4: 268.
64. Ekmekci A, Gungor B, Uluganyan M, Ozcan KS, Bozbay M, et al. Presence of Metabolic Syndrome is not an Independent Predictor of In-hospital Adverse Events in Patients with ST Elevation Myocardial Infarction that Underwent Primary Percutaneous Coronary Intervention. *Endocrinol Metab Synd* 2013; 2:112.